Alerts, Notices, and Case Reports

McCune-Albright Syndrome

A Case of Primary Hypogonadism Obscured by Hyperprolactinemic Hypogonadotropic Hypogonadism

ARTHUR L. M. SWISLOCKI, MD
CARLOS A. CAMARGO, MD
ANDREW R. HOFFMAN, MD
Palo Alto, California

IN THE 50 YEARS since Fuller Albright and his colleagues described a syndrome characterized by "osteitis fibrosa disseminata, areas of pigmentation, and precocious puberty in females,"[1] various case reports and review articles have appeared expanding the endocrinologic dimensions of what has become known as the McCune-Albright syndrome.[2-20] It is now recognized that the syndrome occurs in men as well as women and that the endocrine abnormalities can be manifested as deficiency syndromes and hyperfunctional states. We report the case of a patient with the McCune-Albright syndrome in whom primary gonadal failure was obscured by hypogonadotropic hypogonadism associated with hyperprolactinemia and acromegaly.

Report of a Case

The patient, a 26-year-old man, was the sole product of a nonconsanguineous marriage and a term pregnancy. He had many limb fractures as a child, and a diagnosis of polyostotic fibrous dysplasia was made when he was 5. He underwent the placement of rods in both hips. Puberty began at age 12. Two months before our initial evaluation, a carpal tunnel syndrome developed in his right hand and he noticed a weight gain of 4.5 to 7 kg (10 to 15 lb). There was no change in bowel habits, heat or cold intolerance, or changes in his skin or hair. An episode of nephrolithiasis at age 22 required a lithotomy. The family history was pertinent only for a cousin whose facial features were reportedly similar to those of the patient. He complained of hyposmia and infrequent erections.

On physical examination the patient appeared well developed, virilized, and stocky with leonine facies. He had large pigmented macules with ragged edges on the face, back, and trunk. The thyroid was slightly enlarged and contained a 0.5-cm nodule in the left lobe. There was bilateral Tanner stage II gynecomastia without galactorrhea, a normal-sized circumcised penis, and the testes measured 12 cm^3 bilaterally. The hair distribution was normal for an adult man. He was hyposmic in response to challenge with tobacco, coffee grounds, and tea leaves. The reproductive hormone values were as follows: total testosterone content, 3.85 nmol per liter (normal, 10.4 to 34.7); luteinizing hormone (LH), 6 IU per liter (normal, 4 to 18); follicle-stimulating hormone (FSH), 12 IU per liter (normal, 5 to 20); and prolactin, 120 μg per liter (normal <20). The growth hormone level was 10 μg per liter, and the insulinlike growth factor I-somatomedin C (IGF-I) level was elevated at 519 ng per ml (normal, 40 to 170). A serum growth hormone-releasing hormone level was undetectable. Thyroxine, thyroid-stimulating hormone, iron, and serum iron-binding capacity values were all normal.

Serum growth hormone concentrations did not suppress after a 75-gram glucose challenge, consistent with a diagnosis of acromegaly. There was no gonadotropin response to a 100-μg intravenous challenge of gonadotropin-releasing hormone (GnRH [gonadorelin hydrochloride]).

Radiographs of the legs revealed polyostotic fibrous dysplasia with its characteristic ground-glass appearance. A

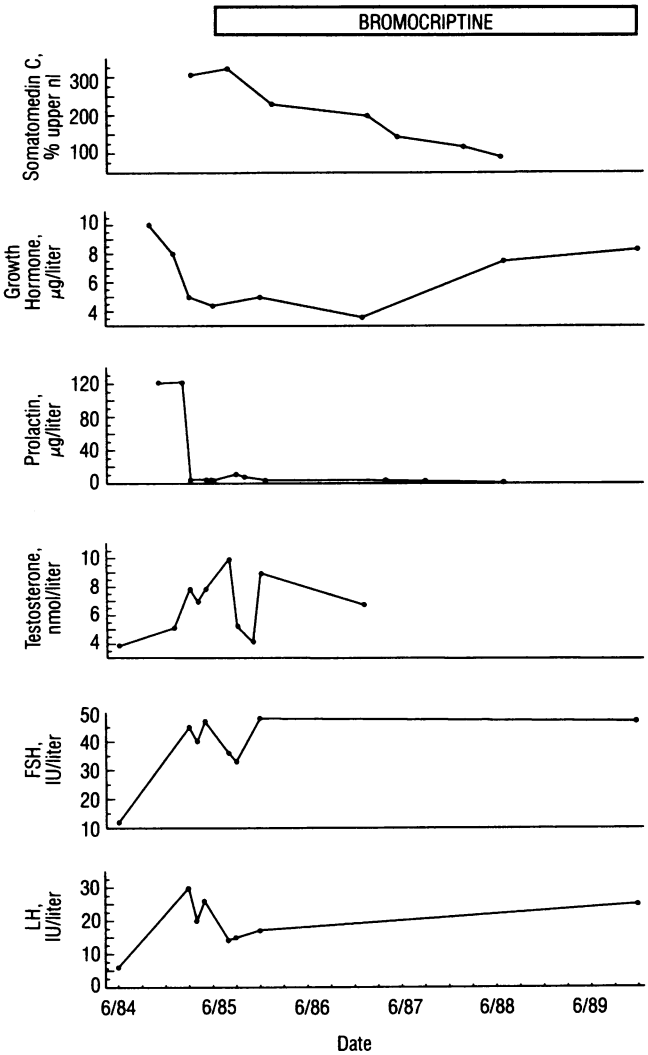

Figure 1.—The clinical response to bromocriptine mesylate therapy is shown. Somatomedin C is expressed as a percentage of the upper limit of normal values; normal values for the other hormones: growth hormone, less than 10 μg per liter; prolactin, less than 20 μg per liter; luteinizing hormone (LH), 4 to 18 IU per liter; follicle-stimulating hormone (FSH), 5 to 20 IU per liter; testosterone, 10.4 to 34.7 nmol per liter. The patient was also receiving testosterone, 300 mg intramuscularly every 3 weeks, between December 1985 and November 1989.

(Swislocki ALM, Camargo CA, Hoffman AR: McCune-Albright syndrome—A case of primary hypogonadism obscured by hyperprolactinemic hypogonadotropic hypogonadism. West J Med 1990 Dec; 153:653-656)

From the Geriatric Research, Education, and Clinical Center, Veterans Administration Medical Center, Palo Alto, and the Division of Endocrinology, Department of Medicine, Stanford University School of Medicine, Stanford, California. Dr Swislocki is currently with the Medical Service, Veterans Administration Medical Center, Martinez, California, and the Division of Endocrinology, Department of Medicine, University of California, Davis.

Dr Swislocki is the recipient of an Individual National Research Service Award grant from the National Institutes of Health, DK 07832-01.

Reprint requests to Arthur L. M. Swislocki, MD, Medical Service (111E), VA Medical Center, 150 Muir Rd, Martinez, CA 94553.

ABBREVIATIONS USED IN TEXT

FSH = follicle-stimulating hormone
GnRH = gonadotropin-releasing hormone
IGF-I = insulinlike growth factor I
LH = luteinizing hormone

computed tomographic scan of the head showed pronounced thickening of the bones of the skull and sella turcica, without any apparent pituitary tumor or erosion of the sella. No abnormality was noted in the area of the pituitary stalk or hypothalamus.

The diagnosis of McCune-Albright syndrome was made, manifesting as polyostotic fibrous dysplasia, characteristic cutaneous "coast of Maine" pigmented lesions,[3] hyperprolactinemia with hypogonadotropic hypogonadism, acromegaly, and euthyroid multinodular goiter. Because the dense bony lesions in the sella turcica might complicate a transsphenoidal operation and because imaging studies failed to show a pituitary tumor, he was treated with bromocriptine mesylate. The clinical response is shown in Figure 1. With the initiation of bromocriptine therapy, there was a fall in the prolactin level and, eventually, of growth hormone levels, with a rise in both gonadotropin and testosterone values. The patient reported that his sense of well-being and libido increased markedly, and he grew a beard. Although subjective improvement persisted, the testosterone concentration again fell below the normal range; the LH and FSH concentrations rose, the LH level to 30 IU per liter and then stabilizing at 17 IU per liter and the FSH level to 45 IU per liter, stabilizing at 35 to 45 IU per liter, suggesting the diagnosis of primary gonadal failure. The patient is currently receiving bromocriptine (10 mg orally three times a day) and testosterone enanthate (300 mg intramuscularly every three weeks). His most recent IGF-I value is 160 ng per ml, but growth hormone and prolactin levels are both within normal limits at 8.3 μg per liter and 2 μg per liter, respectively.

After about four years on a regimen of bromocriptine and testosterone, the patient's radiographic and endocrinologic studies were repeated. Magnetic resonance imaging revealed a 5-mm microadenoma of the pituitary gland. A GnRH stimulation test was done after testosterone therapy had been discontinued for four weeks: 15 to 45 minutes after 100 μg of GnRH was administered intravenously, the FSH level rose from 47 IU per liter at baseline (normal <20) to 63 to 68 IU per liter, and the LH level rose from 25 IU per liter at baseline (normal <15) to 60 to 69 IU per liter. In view of the microadenoma, a neurosurgical consultation was obtained and a decision was made in consideration of the thick bone in the sellar floor to avoid a neurosurgical approach to the pituitary and to treat the patient with stereotactic external beam irradiation. Treatment is pending at this time.

Discussion

The initial definition of the McCune-Albright syndrome required the triad of pigmented skin lesions, fibrous dysplasia of bone, and precocious puberty in girls.[1] Most of the reported cases subsequently grouped under the heading of the McCune-Albright syndrome have included patients with precocious puberty[1-8,11,13-15,17]; there have also been cases of secondary hypogonadism in both sexes.[13,16,17,20] Acromegaly and gigantism have also been reported numerous times.[13,17,19,21-26] The endocrinopathies reportedly associated with the McCune-Albright syndrome are summarized in Table 1.

The case presented in this report is unusual, however, in that the primary hypogonadism was masked by hyperprolactinemia.[27,28] The elevated gonadotropin levels characteristic of primary gonadal failure were initially suppressed. When prolactin secretion was suppressed by bromocriptine therapy, LH and FSH concentrations were disinhibited and rose towards the castrating range.

The primary hypogonadism reported in this patient appears to be unique in the overall experience with this curious syndrome. In the other cases of the McCune-Albright syndrome in which hypogonadism was reported,[13,16,17,20] data were most consistent with secondary (central) hypo-

TABLE 1.—*Endocrinologic Syndromes in the McCune-Albright Syndrome*

Endocrinopathy	*Reference*
Precocious puberty	
Girls	Albright et al, 1937[1]; Benedict, 1962[2]; Danon et al, 1975[5]; D'Armiento et al, 1983[6]; Foster et al, 1984[7]; Chung et al, 1983[13]; Andrews et al, 1974[15]; Lipson and Hsu, 1981[17]; Geffner et al, 1987[25]
Boys	Benedict, 1962[2]; Benedict, 1966[4]; Lightner et al, 1975[8]; Benjamin and McRoberts, 1973[11]; Nitzan et al, 1973[14]
Enlarged testes with Leydig cell hyperplasia	Benjamin and McRoberts, 1973[11]
Hypogonadism (male)	Joishy and Morrow, 1976[20]
Small testes (without precocious or delayed puberty)	Benedict, 1962[2]
Thyroid nodules	Benedict, 1962[2]; Benjamin and McRoberts, 1973[11]
Hyperthyroidism	Benedict, 1962[2]; D'Armiento et al, 1983[6]; Moldawer and Rabin, 1966[10]; Andrews et al, 1974[15]
Acromegaly, gigantism	Lightner et al, 1975[8]; Nakagawa et al, 1985[9]; Chung et al, 1983[13]; Lipson and Hsu, 1981[17]; Harris, 1985[19]; Joishy and Morrow, 1976[20]; Powell, 1976[21]; Lightner and Winter, 1981[22]; Polychronakos et al, 1982[23]; Cuttler, 1986[24]; Geffner et al, 1987[25]; Pacini et al, 1987[26]
Cushing's syndrome	Danon et al, 1975[5]; Benjamin and McRoberts, 1973[11]
Hyperprolactinemia or galactorrhea	Chung et al, 1983[13]; Harris, 1985[19]; Polychronakos et al, 1982[23]; Cuttler et al, 1986[24]; Pacini et al, 1987[26]
Amenorrhea	
Primary	Shires et al, 1979[16]
Secondary	Chung et al, 1983[13]; Lipson and Hsu, 1981[17]
Non-insulin-dependent diabetes mellitus	Moldawer and Rabin, 1966[10]; Lipson and Hsu, 1981[17]
Hyperplastic parathyroidism (or hyperparathyroidism)	Benedict, 1962[2]; Ehrig and Wilson, 1972[12]
Hypophosphatemia	McArthur et al, 1979[18]
Rickets	McArthur et al, 1979[18]
Gynecomastia	Benjamin and McRoberts, 1973[11]

gonadism. The persistently elevated FSH level, the elevated LH levels following the cessation of testosterone replacement, and the exaggerated gonadotropin response to GnRH administration support the diagnosis of primary hypogonadism and rule out the presence of central or hypogonadotropic hypogonadism.

Primary hypogonadism can be developmental or acquired.[27-29] Developmental causes include Klinefelter's syndrome or its mosaic variants. Klinefelter's syndrome is characterized by a 47,XXY karyotype, and the most common variant is a 46,XY/47,XXY mosaic. These patients typically have small, firm testes, impaired sexual maturation, gynecomastia, underandrogenization, and mild mental deficiency. Although the patient reported here has small, firm testes, he has above-normal intelligence, had a normal puberty, and is well androgenized, making Klinefelter's syndrome or one of its variants unlikely.

A more likely explanation is that this patient's primary hypogonadism is acquired. The major causes include infection, trauma, irradiation, and drugs.[29] Respectively, he has no history of mumps orchitis, trauma, or radiation exposure. The typical drugs associated with testicular suppression include cyproterone, spironolactone, and ethanol. This man drinks socially, but there is no evidence by history of impaired job or personal function and no physical evidence or laboratory data to suggest alcohol abuse. He has never received antineoplastic agents or cimetidine, and he says he does not use marijuana.

Other rarer causes of testicular failure include autoimmune orchitis (usually in the context of multiple autoimmune endocrinopathies) and granulomatous disease—specifically, leprosy.[29] Testicular atrophy develops in about 10% to 20% of persons with leprosy, although this is less common in other forms of systemic granulomatous disease. This patient has no evidence of either autoimmune endocrinopathy or granulomatous disease. Thus, although he does not appear to have a developmental cause of testicular failure, there is no clear evidence leading to a cause of acquired testicular failure.

The means by which hyperprolactinemia induces hypogonadism are not well understood. Carter and co-workers pointed out in their series of hypogonadal men with prolactin-secreting tumors that both the testes and pituitary gland responded appropriately to stimulation by human chorionic gonadotropin and exogenous GnRH, respectively, suggesting that GnRH deficiency was a major causative factor.[30] Prolactin may also interfere with the conversion of testosterone to dihydrotestosterone.[31,32] Zini and colleagues reported that hyperprolactinemic men not only had normal basal LH levels in the presence of lower testosterone concentrations but also a delayed peak response of LH levels to GnRH that partially corrected with the administration of bromocriptine.[33]

The initial finding of hypogonadotropic hypogonadism probably reflects suppression of the hypothalamic-hypophyseal axis with a reduction in hypothalamic GnRH secretion by elevated circulating prolactin levels. With the control of hyperprolactinemia by bromocriptine therapy, gonadotropin secretion initially increased, but serum testosterone levels did not reach the normal range, indicating the presence of primary testicular failure.

The cause of the hyperprolactinemia, as well as the hypersomatotropinemia, is most likely the microadenoma uncovered on the recent magnetic resonance imaging scan. Pituitary tumors may produce both growth hormone and prolactin,[34] and in the absence of immunohistochemical confirmation of the tumor tissue type, we can only speculate that this is the explanation. Alternatively, the tumor is producing growth hormone, and prolactin is being released as a result of the tumor interfering with the normal inhibition of prolactin secretion, although the small size of the tumor makes this less likely. A recent examination of coexistent growth hormone and prolactin hypersecretion in three patients with the McCune-Albright syndrome without pituitary masses on high-resolution imaging suggests that abnormal hypothalamic function with or without a concomitant embryologic defect in pituitary development and activity may underlie the disordered secretion dynamics of growth hormone and prolactin.[35]

The combination of anosmia and hypogonadism is occasionally seen in patients with hypothalamic hypogonadism (Kallmann's syndrome). In our patient, however, the hyposmia was probably due to severe sclerotic cranial bone disease obliterating the cribriform plate of the ethmoid and destroying the olfactory filaments.

The great diversity in the presentation of the McCune-Albright syndrome, with an unusual bone disease, skin lesions, and a broad panoply of endocrine dysfunction, suggests that there is a basic defect of regulation of cellular function and growth. The nature of the genetic defect remains unknown, but speculation has centered on an altered regulation of intracellular cyclic adenosine monophosphate action.[36] It has recently been shown that patients with bony lesions and pseudohypoparathyroidism (Albright's hereditary osteodystrophy) have reduced stimulatory G protein in many cells, resulting in the diminished generation of hormone-stimulated cyclic adenosine monophosphate.[37]

REFERENCES

1. Albright F, Butler AM, Hampton AO, Smith P: Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females. N Engl J Med 1937; 216:727-746

2. Benedict PH: Endocrine features in Albright's syndrome (fibrous dysplasia of bone). Metabolism 1962; 11:30-45

3. Benedict PH, Szabo G, Fitzpatrick TB, Siness SJ: Melanotic macules in Albright's syndrome and in neurofibromatosis. JAMA 1968; 205:616-618

4. Benedict PH: Sex precocity and polyostotic fibrous dysplasia. Am J Dis Child 1966; 111:426-429

5. Danon M, Robboy SH, Kim S, Scully R, Crawford JD: Cushing syndrome, sexual precocity, and polyostotic fibrous dysplasia (Albright syndrome) in infancy. J Pediatr 1975; 87:917-921

6. D'Armiento M, Reda G, Camagna A, Tardella L: McCune-Albright syndrome: Evidence for autonomous multiendocrine hyperfunction. J Pediatr 1983; 102:584-586

7. Foster C, Ross JL, Shawker T, et al: Absence of pubertal gonadotropin secretion in girls with McCune-Albright syndrome. J Clin Endocrinol Metab 1984; 58:1161-1165

8. Lightner ES, Penny R, Frasier SD: Growth hormone excess and sexual precocity in polyostotic fibrous dysplasia (McCune-Albright syndrome): Evidence for abnormal hypothalamic function. J Pediatr 1975; 87:922-927

9. Nakagawa H, Nagasaka A, Sugiura T, et al: Gigantism associated with McCune-Albright's syndrome. Horm Metab Res 1985; 17:522-527

10. Moldawer M, Rabin ER: Polyostotic fibrous dysplasia with thyrotoxicosis. Arch Intern Med 1966; 118:379-384

11. Benjamin DR, McRoberts JW: Polyostotic fibrous dysplasia associated with Cushing syndrome. Arch Pathol 1973; 96:175-178

12. Ehrig U, Wilson DR: Fibrous dysplasia of bone and primary hyperparathyroidism. Ann Intern Med 1972; 77:234-238

13. Chung KF, Alaghband-Zadeh J, Guz A: Acromegaly and hyperprolactinemia in McCune-Albright syndrome—Evidence of hypothalamic dysfunction. Am J Dis Child 1983; 137:134-136

14. Nitzan M, Laron Z, Pertzelan A, Scharf A: McCune-Albright syndrome with sexual precocity in a boy. Helv Paediatr Acta 1973; 28:61-65

15. Andrews BS, Posen S, Podolsky S: Thyrotrophin in Albright's syndrome with hyperthyroidism. Ann Intern Med 1974; 81:561

16. Shires R, Whyte MP, Avioli LV: Idiopathic hypothalamic hypogonadotropic hypogonadism with polyostotic fibrous dysplasia. Arch Intern Med 1979; 139:1187-1189

17. Lipson A, Hsu TH: The Albright syndrome associated with acromegaly: Report of a case and review of the literature. Johns Hopkins Med J 1981; 149:10-14

18. McArthur RG, Hayles AB, Lambert PW: Albright's syndrome with rickets. Mayo Clin Proc 1979; 54:313-320

19. Harris RI: Polyostotic fibrous dysplasia with acromegaly. Am J Med 1985; 78:539-542

20. Joishy SK, Morrow LB: McCune-Albright syndrome associated with a functioning pituitary chromophobe adenoma. J Pediatr 1976; 89:73-75

21. Powell DGB: Polyostotic fibrous dysplasia with acromegaly (Albright's syndrome). South Afr Med J 1976; 50:182-183

22. Lightner ES, Winter JSD: Treatment of juvenile acromegaly with bromocriptine. J Pediatr 1981; 98:494-496

23. Polychronakos C, Tsoukas G, Ducharme JR, Letarte J, Collu R: Gigantism and hyperprolactinemia in polyostotic fibrous dysplasia (McCune-Albright syndrome). J Endocrinol Invest 1982; 5:323-326

24. Cuttler L, Levitsky LL, Zafar MS, Mellinger RC, Frohman LA: Hypersecretion of growth hormone and prolactin in McCune-Albright syndrome (Abstr). Pediatr Res 1986; 20:213A

25. Geffner ME, Nagel RA, Dietrich RB, Kaplan SA: Treatment of acromegaly with a somatostatin analog in a patient with McCune-Albright syndrome. J Pediatr 1987; 111:740-743

26. Pacini F, Perri G, Bagnolesi P, Cilotti A, Pinchera A: McCune-Albright syndrome with gigantism and hyperprolactinemia. J Endocrinol Invest 1987; 10:417-420

27. Franks S: Male reproductive endocrinology. Clin Obstet Gynaecol 1980; 8:549-569

28. Wu FCW: Male hypogonadism—Current concepts and trends. Clin Obstet Gynaecol 1985; 12:531-555

29. Griffin JE, Wilson JD: Disorders of the testes and male reproductive tract, *In* Wilson JD, Foster DW (Eds): Williams Textbook of Endocrinology, 7th edition. Philadelphia, Pa, WB Saunders, 1985, pp 259-311

30. Carter JN, Tyson JE, Tolis G, Van Vliet S, Faiman C, Friesen HG: Prolactin-secreting tumors and hypogonadism in 22 men. N Engl J Med 1978; 299:847-852

31. Mawhinney MG, Belis JA, Thomas JA, et al: Effect of prolactin on androgen metabolism by the guinea-pig sex accessory organs. J Pharmacol Exp Ther 1975; 192:242-249

32. Magrini G, Ebiner JR, Burckhardt P, et al: Study on the relationship between plasma prolactin levels and androgen metabolism in man. J Clin Endocrinol Metab 1976; 43:944-947

33. Zini D, Carani C, Baldini A, Cavicchioli C, Piccinini D, Marrama P: Further acquisitions on gonadal function in bromocriptine treated hyperprolactinemic male patients. Pharmacol Res Commun 1986; 18:601-609

34. Baumann G: Acromegaly. Endocrinol Metab Clin 1987; 16:685-703

35. Cuttler L, Jackson JA, Saeed uz-Zafer M, Levitsky LL, Mellinger RC, Frohman LA: Hypersecretion of growth hormone and prolactin in McCune-Albright syndrome. J Clin Endocrinol Metab 1989; 68:1148-1154

36. Lee PA, Van Dop C, Migeon CJ: McCune-Albright syndrome: Long-term follow-up. JAMA 1986; 256:2980-2984

37. Levine MA, Jap TS, Mauseth RS, Downs RW, Spiegel AM: Activity of the stimulatory guanine nucleotide-binding protein is reduced in erythrocytes from patients with pseudohypoparathyroidism and pseudopseudohypoparathyroidism: Biochemical, endocrine, and genetic analysis of Albright's hereditary osteodystrophy in six kindreds. J Clin Endocrinol Metab 1986; 62:497-502

Revenge of the Barbecue Grill
Carbon Monoxide Poisoning

JOHN D. GASMAN, MD
JOSEPH VARON, MD
JAMES P. GARDNER, MD
Stanford, California

CARBON MONOXIDE (CO) poisoning can be difficult to recognize because it often has a presentation that suggests a viral illness, food poisoning, gastroenteritis, or even a functional illness. Because CO is the leading cause of death due to poisoning in the United States,[1-3] emergency department physicians should suspect CO poisoning when a similar illness occurs simultaneously in two or more persons.

Report of Cases

Four members of a non-English-speaking family presented to a university hospital emergency department, all complaining of headache, lethargy, abdominal cramping, and nausea. They related a history of drinking unrefrigerated milk the evening before presentation. Two patients had mild orthostatic blood pressure changes. They were treated with intravenous fluids and discharged with the diagnosis of infectious gastroenteritis. One family member remained in the emergency department with persistent symptoms.

Several hours later, four other members of the same family presented to the emergency department with similar complaints. They were evaluated and treated with intravenous fluids, and all were discharged, including the patient from group 1. Several hours later, four more members of the same family presented to the emergency department with similar complaints. One patient had not ingested the suspected milk, but she suffered from migraine headache and thought that this was one of her headaches.

Further questioning through an interpreter revealed that all patients were members of the same household. Specific questions about home heating systems did not suggest mechanical problems; in fact, it had not been used. An unaffected relative, who lived elsewhere, admitted to cooking indoors with charcoal briquets in the fireplace after originally stating that the cooking was done outdoors.

Specimens were drawn for arterial blood gas determinations and carboxyhemoglobin (COHb) concentrations. Results were 0.069, 0.151, 0.171 and 0.174 (normal < 0.150).* The four patients in the emergency department were treated with 100% oxygen for two hours with symptomatic improvement. The COHb concentration was rechecked in the patient who had a 0.174 level, and it had fallen to 0.044 (4.4%). Fire fighters were dispatched to check the house and reported a barbecue grill in the middle of the living room and evidence of smoke damage.

Discussion

Carbon monoxide is the leading cause of death due to poisoning in the United States.[1-3] It is also the most common cause of death in combustion-related inhalation injury. Claude Bernard in 1857 provided the first accurate description of CO poisoning. Since then many advances in our understanding of the pathophysiology have been achieved.

Carbon monoxide combines preferentially with hemoglobin to produce COHb, displacing oxygen and reducing the systemic arterial oxygen content. Possible mechanisms of toxicity include a decrease in the oxygen-carrying capacity of blood, alteration of the dissociation characteristics of oxyhemoglobin so that delivery to the tissues further decreases, and a decrease in cellular respiration by binding with cytochrome a_3.

Goldbaum and co-workers postulated that high dissolved CO concentrations develop only at the alveolar-blood interface when CO is inhaled. Dissolved CO delivered to tissues, especially heart and brain, causes most of the CO toxicity by binding with the cytochrome a_3.[4] Hence, it is dissolved CO, not COHb, that causes toxic reactions.

The half-life of COHb is 320 minutes for young healthy volunteers breathing room air. Administering 100% oxygen at 1 atm reduces the half-life to 80.3 minutes, whereas 100% oxygen at 3 atm will reduce the half-life to 23.3 minutes.[5]

Carbon monoxide binds to cardiac and skeletal myoglobin and hemoglobin. Cardiac myoglobin binds three times more CO than skeletal myoglobin.[6] Because carboxy-

(Gasman JD, Varon J, Gardner JP: Revenge of the barbecue grill—Carbon monoxide poisoning. West J Med 1990 Dec; 153:656-657)

From the Department of Medicine, Stanford University Medical Center (Drs Gasman and Varon), and the Division of Emergency Medicine, Department of Surgery, Stanford University School of Medicine (Dr Gardner), Stanford, California.

Reprint requests to John D. Gasman, MD, Department of Medicine S-102D, Stanford University Medical Center, 300 Pasteur Dr, Stanford, CA 94305.

*In Système International units, these values are expressed as a fraction of 1. In conventional units, they would be 6.9%, 15.1%, 17.1%, and 17.4%.